

the hypothesis that α_2 agents affect frontal lobe functioning, and provides a rationale for the pharmacological treatment of frontal dementias. This preliminary report in a single patient awaits extension to a larger sample.

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Ictal language shift in a polyglot

There are a number of dysfunctions of language associated with epilepsy. During an episode usually associated with a temporal lobe focus patients may have loss of speech,¹ dysphasia,² or automatisms.³ A patient is described who speaks six languages and has automatisms in a number of these languages. She is a 49-year-old Indian woman with no history of major illness, loss of consciousness, or head trauma. The episodes first began in 1980. She was speaking English and suddenly spoke a few words of Gujarati that were totally out of context. She did not remember what she said and had no change of consciousness associated with this. The patient was unwell at the time with a urinary tract infection. The second episode was in 1991, when she had an episode during which she shifted from Punjabi to English but did not remember the context. There have been four further episodes in the past year. On one occasion she was on a telephone speaking to a sister in Punjabi and said in Gujarati "I don't know, there is nothing we have cooked." She does not remember anything about this conversation but remembered what she had said previously and carried on with further conversation normally. On another occasion she again spoke in Gujarati "we don't have clothes". On a further occasion she switched from Gujarati to Urdu, saying "the closet is empty, there is nothing in the closet." The conversation was continued for a short time by the sister but the patient could not remember this. The last episode was while teaching in English, she spoke Punjabi.

Some of these episodes have been

observed by her mother who is fluent in all these languages and noticed that the patient had flickering eyes and gulping.

The patient speaks Punjabi at home and speaks English and Swahili commonly. She also learnt Gujarati as a child, is able to read and write Hindi, and speaks Urdu.

On examination the patient was alert and orientated. There were no abnormalities on neurological examination. An EEG showed bilateral temporal lobe spikes, more on the right, which were accentuated with over-breathing. A CT scan was normal. The patient was given 100 mg carbamazepine twice a day and no further episodes occurred during the ensuing four months.

A variety of speech disturbances may occur in patients with temporal lobe epilepsy. Dysphasia is associated with seizures originating in the dominant hemisphere.^{1,3} Investigations to localise the disturbances have been made by stimulation studies.^{3,4} Speech automatisms have usually been localised to the temporal lobe of the non-dominant hemisphere, although they are occasionally noted in the dominant hemispheres.¹

Much information exists on the speech disturbances in multilingual people with strokes. There are two main themes, one propounded by Ribot, who said that the first language to be acquired is the first to recover and one by Pitres, who proposed that the language most familiar and most often used, will return first.² The emotional attachment to a language is also important.² Recovery may be parallel, when all the languages recover at a similar rate, which is the most common, differential, or successive. The patient may even develop a foreign accent after a cerebral infarction. In a study of bilingual patients given stimulation there can be a dissociation of sites where naming is affected.⁴ There are only rare reports of language shift in epileptic patients. In a patient undergoing stimulation of the right temporal lobe,³ the patient said "je suis malade" later followed by "I seem to lose my memory". Another patient, who was a Polish and English speaker, had as a seizure phenomenon the utterance "I beg your pardon" which occurred during a Polish conversation; this was also provoked by metrazol.¹

The patient described here had speech automatism in a variety of Asian languages and English. Cases have been reported of language problems in Chinese-English speakers with strokes but reports for Indian-English speakers are rare. One patient spoke Telugu, Kannada, Tamil, and English fluently, but after a stroke had pure alexia in Kannada and English.⁵

The mechanism for shift of languages is not clearly understood. It has been postulated that it is due to a lesion of the supramarginal gyrus, but this holds true in only rare cases. The switching of languages is probably more of a generalised than specific neural mechanism.² Automatisms, as in our patient, are more localised to the temporal lobes and usually the non-dominant hemisphere.

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Relief of trigeminal neuralgia by proparacaine

Two recent publications^{1,2} have reported the successful treatment of trigeminal neuralgia by the ophthalmic anaesthetic proparacaine hydrochloride 0.5% instilled in the eye of the affected side.

We tried this medication in 15 patients suffering from neuralgia involving one or two branches of the trigeminal nerve. In three patients there was involvement of the second division of the nerve, whereas in eight, the first and second divisions were affected. The other four patients complained of pain in the distribution of the second and third divisions of the nerve. In 13 patients the neuralgia was idiopathic; all had been taking carbamazepine for a considerable time with partial or no response. One patient had a giant suprasellar aneurysm and another a large acoustic neuroma, both inoperable because of the patients' advanced age and general condition.

The treatment consisted of instillation of two drops of proparacaine hydrochloride 0.5% in the eye of the affected side, in every case. A satisfactory effect was obtained in 13 patients with a clear cut improvement of symptoms allowing withdrawal or reduction of the daily dose of carbamazepine. In two patients (including a hospital physician) the medication stopped an attack of severe pain and this result is now permanent. Instillation was repeated once in eight patients and twice in two patients to obtain a stable and lasting result. The observation period ranged between one and four months and no side effects were reported by the patients. One patient experienced no change in her symptoms after the initial instillation and refused a second attempt, and another patient was lost to follow up.

In accordance with the previous authors, we find it difficult to propose a mechanism by which a benzoic ester with topical anaesthetic effect produces lasting relief of neuralgia in the distribution of the divisions of the trigeminal nerve that differ from that on which the drug acts directly. Is it possible that some of the drug drained through the lacrimal duct in the nasal and oral cavities, was absorbed by the mucosa and the nerve endings of the second division of the nerve, and eventually reached the trigeminal nucleus via retrograde axonal transport?

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